Comments on the Draft Environmental Impact Report for Chlorinated Polyvinyl Chloride (CPVC) Pipe Use for Potable Water Piping in Residential Buildings

Final Report

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Introduction

The purpose of this document is to present a critique of the adequacy, accuracy and completeness of the toxicological and epidemiological issues evaluated in the Draft Environmental Impact Report (draft EIR) for chlorinated polyvinyl chloride (CPVC) pipe use for potable water piping in residential buildings. After review of the draft EIR and the relevant literature, we find that the conclusion that use of CPVC pipe for potable water piping in residential buildings will not result in any significant adverse impacts on the environment is not supported by the analysis presented in the draft EIR or its technical appendix. We also find evaluation of the potential health effects to exposed workers and residents is deficient and at times inaccurate. Based on the analysis which follows, we find that significant adverse health effects on workers and drinking water consumers from the proposed CPVC approval would not be unexpected.

The following discussion presents our detailed evaluation of the draft EIR. The first section presents a general outline of some of the criticisms of the report. This is followed by a detailed discussion of the sections of the document pertaining to potential health effects and environmental impacts.

General Outline of Comments

The draft EIR on CPVC pipe use for potable water is deficient in several toxicological aspects. In terms of general population health risks, the draft EIR has failed to consider:

- that the NSF standards on which its health risk comparison is based do not appear to be health protective
- the possibility of synergistic or additive effects between different chemicals in CPVC pipe leachate and/or common contaminants of tap water
- exposure from inhalation and dermal absorption
- the possible carcinogenicity of THF
- the potential toxic effects resulting from exposure to mono and diorganotins
- the lack of long term exposure data for MEK and acetone, and the potential developmental and reproductive effects of these substances

In terms of worker health issues, the draft EIR has failed to adequately consider:

- that the TLV's for worker exposure to THF, MEK, acetone and several other solvents may not be adequately health protective
- that combined dermal and inhalation exposure to CPVC cement solvents and primers may result in significant adverse short term and long term health effects
- the potential carcinogenicity of THF
- the lack of chronic toxicity data for MEK
- potential interactions of acetone, MEK, THF and solvent mixtures
- the fact that short term exposures to these solvents may produce chronic health effects
- a worst-case analysis analysis of worker exposure

With regards to the analysis of the effects of existing piping materials and their effect on the general population, workers, and the environment, the draft EIR does NOT provide data to substantiate the assertions that:

- exposure to copper pipe and steel pipe leachate would result in adverse health effects to drinking water consumers
- workers exposed to copper pipe solders and fluxes are in danger of adverse health effects
- the environmental impacts of copper pipe are greater than those resulting from CPVC pipe

SPECIFIC COMMENTS

NSF Standards for CPVC Related Solvents and Organotins in Drinking Water

The draft EIR states that it has relied on Maximum Allowable Limits (MAL's) determined by the National Sanitary Foundation (NSF) for CPVC related solvents and organotins since such standards have not been established by the USEPA or the California Department of Health Services. The MAL's were said to be calculated using methodology as described in ANS/NSF 61 (1997) Appendix A which includes two sets of standards, Maximum Drinking Water Levels (MDWL's) and Short Term Exposure Levels (STEL's). It should be noted that the methodology for calculating the MAL's as described in this appendix does not account for multipathway exposures, additive or synergistic effects, or exposure of infants and children.

The NSF STEL's relied upon by the draft EIR are not adequately defined in the draft EIR nor in NSF61, Appendix A. Note that the American Conference of Governmental

Industrial Hygienists (ACGIH) defines a STEL as an airborne concentration of a substance that represents conditions under which it is believed that nearly all workers may be exposed for a short period of time (usually 15-minutes) without adverse affect (Klaassen, 1996). For purposes of our commentary, we assume that the STEL's refer to acute or subacute exposure. Nonetheless, it is not possible to conduct a complete and meaningful evaluation of the adequacy of the action levels without a more complete description (i.e., do the STEL's pertain to a 1-hour, 1-day, 1-week, several weeks exposure?).

In addition, the draft EIR must describe the underlying toxicological or epidemiological studies and data upon which each of the MAL's (MDWL's and STEL's) are based. Meaningful conclusions regarding the extent to which the NSF's MAL's protect public health cannot be made without this information. It is essential in this case that the NSF standards be fully described and the underlying scientific basis for those levels provided since we conclude based on even a brief review of the literature that six of the NSF values are too high to be adequately health-protective.

Before presenting our analyses, the following is a brief discussion of the scientific process and risk assessment methodology by which standards are developed. The toxicological and epidemiological literature for a particular substance is thoroughly reviewed and evaluated. For noncarcinogens, the No Observable Adverse Effect Levels (NOAEL's) or Lowest Observable Adverse Effect Levels (LOAEL's), are identified from the available studies according to exposure pathway and duration. The study which provides the highest NOAEL (or lowest LOAEL if a NOAEL is unavailable) that is also scientifically sound is chosen for the risk assessment.

A key factor in determining the NOAEL or LOAEL is exposure duration (USEPA, 1993). Ideally, data will be obtained from studies with an exposure duration comparable to the exposure duration for which the standard is being derived. For example, 1-day, 10-day, Longer-term and Life-time Health Advisories (HA's) are calculated for substances in drinking water by the USEPA for exposed adults and children (ibid). Each HA corresponds approximately to acute, subacute, several years, and lifetime exposures, respectively. Exposure route is also important. For establishing drinking water standards, the oral route is preferred. When available, human data are chosen over results in laboratory animals.

In order to incorporate a margin of safety, uncertainty factors are applied to the NOAEL or LOAEL (USEPA, 1993). These values, also referred to as safety factors, range from 1 to 10,000 depending on the nature and quality of the data. Uncertainty factors are selected based on scientific judgement and attempt to account for inter and intra-species variability, and any other uncertainty related to the completeness and applicability of the available data. Once the NOAEL or LOAEL is divided by the uncertainty factors, the resulting value, sometimes referred to as a reference dose or acceptable daily intake, is adjusted by the average human body weight (70 kg for adults; 10 kg for children) and water consumption per day (2 liters for adults; 1 liter for children) to arrive at the drinking water standard for a given duration of exposure. These standards are concentration levels at or below which no adverse health effects are anticipated (Lam et al, 1994; USEPA, 1993).

The MDWL Relied Upon by the Draft EIR for Diorganotins is Too High

- Minimum effective and no-observed-adverse-effect doses for dibutyltins were described in the Environmental Health Criteria document on organotins (WHO, 1980). Dibutyltin sulfide administered to rats at oral doses of 1.0, 0.1, 0.01, and 0.001 mg/kg body weight per day for 7 months was tolerated without any detected adverse effects up to a dose of 0.01 mg/kg/day. Higher levels caused intoxication (Mazaev and Korolev, 1969). Using the results of this study and the USEPA risk assessment methodology as described in ANS/NSF 61 Appendix A, we calculate a MDWL of 3.5 ppb versus the NSF standard of 20 ppb for diorganotins. Our number was derived using the No-Observed-Adverse Effect Level (NOAEL) of 0.01 mg/kg/day divided by an uncertainty factor (UF) of 100 (UF of 10 to account for interspecies extrapolation and a UF of 10 to account for intraspecies variability). The resulting value, referred to as a reference dose (RfD), was then multiplied by the assumed human body weight of 70 kg and divided by the average daily intake of drinking water (2 liters per day) to arrive at the MDWL. To protect children, the MDWL would need to be even lower.
- It is important to note that this MDWL refers to an exposure duration of 7 months in rats and not lifetime exposure. In order to account for a human lifetime exposure to diorganotins one would apply another factor of 10 to obtain a lifetime MDWL of 0.35 ppb. This latter value is approximately 57 times lower than the NSF MDWL of 20 ppb.
- The draft EIR states on page 39 that the Lead agency has reviewed the literature regarding organotins leaching from CPVC pipe and found reported values of 10 ppb initially declining to less than 1 ppb after 20 or so extractions. The results of Boettner et al, (1982) indicate that levels of organotins may exceed a MDWL of 0.35 ppb and thereby result in adverse health effects. The draft EIR cannot conclude that there are no significant adverse health effects resulting from long term exposure to organotins in leachate without considering the aformentioned analysis.

<u>The STEL Relied Upon by the Draft EIR for Organotins May Be Too High</u>

- Behavioral studies with monomethyltin have shown that these compounds can induce learning deficiencies in young rats (Noland et al, 1982). Exposure of rat pups to monomethyltin chloride via their dams drinking water throughout gestation and postpartum for 21 days experienced significant learning deficits and changes in locomotor activity levels. Animals in all dose groups (12, 40, 120 mg/l of tin) displayed some learning disability compared with the controls.
- Using the dose of 12 mg/l as the Lowest-Observed-Adverse Effect Level (LOAEL) and the USEPA risk assessment methdology (as described in CH2M Hill, 1987) we calculate a (subacute) short term exposure level of 30 ppb as follows:

  12 mg/l X 25 ml water/day X 1 l/1000 ml = 0.3 mg/rat/day

  -where 12 mg/l is the LOAEL

  -25 ml water/day is the average amount of water consumed by the rat

  -1 l/ 1000 ml converts liters to millilters

  -0.3 mg/rat/day is the estimated dose per rat per day

  Using the dose per rat and weight of the average rat, we convert the dose per rat per day to a dose in milligrams per kilograms of weight per day:

  0.3 mg/rat/day X 1 rat/0.350 kg rat = 0.86 mg/kg/day

- Using an UF of 1000 to account for extrapolation of a LOAEL to a NOAEL, interspecies extrapolation, and intraspecies variability (factor of 10 for each "uncertainty"), the assumed average human weight (70 kg) and the assumed average water consumption per day (2 l/day), we calculate an STEL (subacute) of 30 ppb. This value is approximately three times lower than the NSF standard of 100 ppb.
- Furthermore, Boettner et al (1982) reported initial concentrations of organotins in CPVC pipe leachate that could exceed a STEL of 30 ppb. As a consequence, adverse health effects resulting from subacute exposure to organotins would not be unexpected. The draft EIR cannot conclude that there are no significant adverse health effects resulting from short term exposure to organotins in leachate without considering the aformentioned analysis.

The MAL's Relied Upon by the Draft EIR for MEK and Acetone are Too High

- The USEPA has developed a health advisory for methyl ethyl ketone (MEK) that indicates a long term level for drinking water of 200 ppb (CH_2M Hill, 1987; 1989 draft EIR). This standard is 5 times lower than the MDWL calculated by the NSF.
- The USEPA health advisory for MEK and the NSF MAL's for acetone and MEK are derived and based on exposure to these chemicals individually. As a consequence, these standards will underestimate the potential health risks since their major toxic effects are not due to exposure to these chemicals alone but in combination with other chemicals whose toxicity they potentiate. Hewitt et al (1986) showed that a dose of MEK as small as 0.072 g/kg potentiated the toxic effects of chloroform given 18 hours later. Lower doses were not studied. Using 0.072 g/kg as a LOAEL divided by a UF of 1000 (UF's of 10 to account extrapolation of a LOAEL to a NOAEL, intra and interspecies variability) gives a RfD of 72 ug/kg. Multiplication of the RfD by the assumed human body weight of 70 kg and division by the average daily intake of drinking water (2 liters per day) gives a STEL based on a one time exposure to MEK of 2.5 ppm in the presence of chloroform versus the NSF's STEL of 32 ppm. Note that chloroform is an ubiquitous contaminant of chlorinated drinking water (ATSDR, 1997; CHDS, 1997; Lam et al, 1990) and may also be present in CPVC pipe leachate (Draft EIR, pages 39 and 41). Based on the aforementioned discussion, the draft EIR cannot conclude that the NSF standards would protect against significant adverse health effects resulting from exposure to MEK and acetone without considering potentiated toxic effects.

The NSF has Calculated MAL's for THF Which are Lower Than Those Stated in the Draft EIR

An NSF draft document entitled "Tetrahydrofuran MAL Report" (Whittaker, 1997) describes the calculation of a STEL of 6.26 ppm for THF versus the STEL of 100 ppm cited in the draft EIR. Also, Whittaker (1997) reports a MDWL of 20 ppb for THF versus the level of 1 ppm reported in the document. It is important to note that although these standards are significantly lower than the values reported in the draft EIR, they do not account for multipathway exposures, the potential for synergistic or additive effects, and the exposure of infants and children. In order to properly evaluate the health effects from project THF exposures, the draft EIR must consider the proposed MAL's for THF and must also account for multipathway exposures, synergistic or additive effects, and exposure of infants and children.

Conclusions by the Draft EIR based on the NSF Standards

It is important to note that the draft EIR arrives at its conclusions that CPVC solvent and pipe leachate would not occur at levels that would constitute a significant health impact based on comparison of data from leaching studies with the NSF standards. However, the data from the leaching studies relied upon by the draft EIR are not described in the document or its technical appendix. It is impossible to comment meaningfully on the draft EIR's conclusions without this information. All studies in the peer-reviewed published literature should be included in this comparison. It is important to note that there are few if any published studies that have examined levels of all potential solvent and pipe leachate after long term use of CPVC pipe for potable water.

As demonstrated above, at least six of the NSF standards relied upon by the draft EIR are not health protective. Furthermore, none of the standards account for exposures through multipathways nor synergistic or additive effects. It is also unclear if these levels have accounted for exposure to children as well as adults. Therefore, any conclusions of the draft EIR that the concentrations in leachate are below the threshold of any measureable effects known have not been substantiated. We find that significant adverse health effects would not be unexpected at the current NSF standards.

Finally, the statement on page 21 that "neither USEPA nor the California Department of Health Services have established maximum contaminant levels (MCL's) for solvents used to join CPVC pipe may be due to the relatively low toxicity of these substances" is speculative and should be deleted. In many cases, standards are not developed because the underlying toxicological and/or epidemiological data on which they must be based are not available.

Conclusions of the Draft EIR based on the Risk Assessment Conducted by CH2M Hill

The draft EIR states on page 36 that the Lead Agency previously examined the contamination of drinking water by CPVC related solvents and copper pipe leachate in a comparative health risk assessment conducted under contract by CH2M Hill (1987). This report concluded that the health risks due to CPVC pipe leachate were found to be essentially nonexistent. There are several problems with the risk assessment that makes its conclusions invalid:

- The concentrations of leachate from a 75-day study were compared with Maximum Contaminant Levels or Health Advisories for CPVC solvents. As is the case with the present draft EIR, the standards on which the conclusions are based did not account for multipathway exposures, synergistic and additive effects, and the exposure of infants and children.

- At the time the risk assessment was conducted, the carcinogenicity of THF was unknown. The leaching study depended upon by the risk assessment measured an average high usage concentration of THF in municipal water of 2700 ppb (CH2M Hill, 1987). The average low usage concentration for THF was 810 ppb. These levels of THF are approximately 40 to 135 times higher than the NSF MDWL for THF of 20 ppb. As discussed in the section on NSF standards, the MDWL of 20 ppb may not be adequately health protective.
- Leachate levels and health effects of organotins were not considered in the risk assessment.

Background Levels of Organotins and Solvents Used for CPVC Pipe

The draft EIR's statements that "only one of four solvents (MEK) used in CPVC has been detected in drinking water" (page 20) and that "DHS is not aware of any detection of the organotin stabilizers in the State's drinking water sources" are misleading. According to the "Drinking Water Quality Monitoring Data 1984-1996 Annual Status Report" (CDHS, 1997), only MEK and acetone have been analyzed. Table A-7 in this same document lists initial sampling for chemicals in public drinking water sources according to each year testing has been conducted. Organotins, THF, and cyclohexanone are not included in the testing (ibid). Thus, there is no factual basis to support the draft EIR's suggestion that organotins are not present in California drinking water sources.

Available Information on the Potential Carcinogenic, Reproductive and Developmental Effects of Solvent Origin Leachates

The draft EIR's statement on page 35 that none of CPVC solvents "are classified as human carcinogens by any agency, nor are they known to be reproductive/developmental toxicants" is misleading and is an inaccurate representation of the health effects information available for these chemicals.

- Acetone has not been tested for carcinogencity in experimental animals nor are there data available in the literature regarding its cancer causing potential in humans (ATSDR, 1994). There is some evidence of fetotoxicity, and effects on reproduction and development in exposed animals (ibid). Shortening of the menstrual cycle in humans has also been observed (ibid).

- The International Agency for Research on Cancer (IARC, 1989) has stated that the there is inadequate evidence of the carcinogenicity of cyclohexanone based on one bioassay where slightly elevated tumor incidences were observed at low but not high doses in exposed rats and mice. No prenatal toxicity has been observed in mice exposed to cyclohexanone (ibid). There are no studies available regarding reproductive, developmental, or carcinogenic effects of exposure to cyclohexanone in humans.

- There are no long term studies of the carcinogenicity of MEK in experimental animals (WHO, 1993). The results of epidemiological studies of cancer are inconclusive due to bias in the form of the "Healthy Worker Effect", inadequate follow-up, insufficient power, and/or exposure misclassification (ibid). The total number of litters containing fetuses with anomalous skeletons was significantly increased in rats exposed to MEK by inhalation (Schwetz et al, 1974). It was concluded that MEK is embryotoxic, fetotoxic and potentially teratogenic without inducing maternal toxicity. Deacon et al (1981) found MEK to be slightly fetotoxic with some minor variations in the development of the fetal skeleton. There are no data in the literature concerning the reproductive or developmental effects of exposure to MEK in humans. However, Lowengart et al (1987) conducted a case-control study of children less than 10 years of age to investigate the causes of leukemia. Analysis of information from 123 matched pairs showed an increased risk of leukemia for children whose fathers had occupational exposure after the birth of the child to MEK (OR=3.0).

- There are no studies regarding the carcinogenic, reproductive or developmental effects of THF in humans. Mast et al (1991) evaluated the potential for developmental toxicity in rats and mice exposed to THF by inhalation. The investigators concluded that THF may be embryotoxic in mice, but if the conceptus survives, development as assessed by this experimental design continues in a normal fashion. The embryotoxicity of THF administered to rats on days 6 through 15 of gestation was expressed as developmental delay of the fetus (USEPA/OTS 1992). It has been concluded that there is some evidence in male rats and clear evidence in female mice of the carcinogenicity of THF via inhalation exposure (Chhabra et al 1998; NTP, 1996).

In summary, the fact that MEK and acetone have not been classified as carcinogens by any agency may be due to the scarcity of data. Consequently, it is premature to conclude that long term exposure to these chemicals would not cause carcinogenic effects. Moreover, the few studies that have been conducted indicate that reproductive and developmental effects could result from exposure to MEK and acetone. Recent results from the National

Toxicology Program provide evidence for the carcinogenicity of THF. Mischaracterization and disregard of the results of this study by the draft EIR are discussed in detail in the next section.

Potential Carcinogenicity of THF

The conclusions in the document regarding the National Toxicology Program (NTP) bioassay of the carcinogenicity of THF are misleading and inaccurate. The authors of the draft EIR have improperly dismissed the results and conclusions of the NTP by mischaracterizing standard animal cancer bioasssay protocol, and by ignoring the relevance and importance of the use of these studies in determining the potential for carcinogenicity in humans.

The 2-year NTP inhalation study provided evidence of the carcinogenicity of THF in laboratory animals (Chhabra et al, 1998; NTP, 1996). Renal tubule epithelial adenomas and carcinomas were induced in male rats and were considered to be related to the administration of THF. Although not statistically significant, the combined incidence of adenomas and carcinomas exceeded the historical range for controls in 2-year NTP inhalation studies and occurred with a positive trend in the 600 ppm and 1800 ppm dose groups. Similarly, the incidence and multiplicity of liver neoplasms in female mice were significantly greater than controls in the 1800 ppm dose group. Increases in the 200 ppm and 600 ppm exposure groups were not statistically significant but did show a dose-response relationship. The investigators noted that furan and 1,4- dioxane, chemicals structurally related to THF, have also been shown to cause cancer in rats and guinea pigs (IARC, 1976; NTP, 1993a). The liver was one of the major sites where tumors were induced. Because there is little evidence of its mutagenicity in a variety or *in vitro* and *in vivo* assays, the carcinogenic activity of THF is believed to occur through nongenotoxic modes of action (Chhabra et al, 1998; NTP, 1996). The NTP concluded that based on the results of this study, the Threshold Limit Value (TLV) for THF of 200 ppm is too high since tumors were induced in mice at this dose (exposure of workers installing CPVC pipe to THF will be discussed in detail in a later section).

Animal cancer bioassays are conducted at high doses in an attempt to provide sufficient statistical power to detect a cancer risk among the relatively small number of animals tested (USEPA, 1996). The highest dose chosen is generally one that has been shown to be minimally toxic and is known as the Maximum Tolerated Dose (MTD). Note that most chemicals shown to be carcinogenic in humans have been found in occupational settings where workers were exposed to toxic high doses- a situation similar to the rodent bioassay (Popp, 1984).

A carcinogenic effect at the highest dose may be a consequence of cell killing rather than an inherent carcinogenicity of the substance tested (USEPA, 1996). If sufficient data demonstrate that the effects seen are solely the result of excessive toxicity rather than carcinogenicity of the substance tested, then the effects may be considered as inappropriate to include in an assessment of the potential for human carcinogenicity (ibid). However, this does not appear to be the case in the NTP bioassay of THF. Excessive toxicity was observed in the highest dose group of male mice with a concomitant decrease in survival relative to the controls (Chhabra et al, 1988; NTP, 1996). Neoplastic lesions were not observed in this group of animals relative to the controls. No other manifestations of excessive toxicity were observed in the remaining animals in the study, including those dose groups in which tumors were induced.

The draft EIR statement that THF is not considered to be a human carcinogen is misleading and is contrary to standard public health regulatory practice. As stated in the previous section, there are no studies of the carcinogenicity of THF in humans. If no human data are available, it is assumed that cancer demonstrated in animals indicates that the agent tested can also have carcinogenic potential in humans (USEPA, 1996). The assumption is public health protective and is supported by the fact that nearly all of the agents known to cause cancer in humans do so in animals when adequately tested (IARC, 1994; Tomatis et al, 1989; Huff, 1993). Furthermore, nearly one-third of human carcinogens were identified subsequent to animal testing (Huff, 1993). Conversely, of 82 chemicals for which there is some epidemiological evidence of human carcinogenicity, all but one (arsenic) that have been tested in laboratory animals have been shown to be carcinogenic (Maugh, 1978). Additional support for the relevance of animal data to predict human cancer comes from research on the processes of cancer at the molecular level that show remarkable similarity among species (USEPA, 1996).

There has been much discussion concerning the relevance of the mouse liver tumor to human carcinogenesis (Goodman et al 1991; Popp, 1984). The use of the B6C3F1 mouse has been criticized due to the high rate of spontaneous liver tumor development, particularly in males. Nevertheless, the mouse has been considered a reasonable test animal for hazard identification since its genetic background may act as an amplifier of any tested substances potential carcinogenic activity (Stevenson et al, 1990). If THF were found to cause only liver tumors in mice and no tumors of any type in other species, their significance might be justifiably questioned (USEPA, 1996). However, under the conditions of the NTP bioassay, THF caused a neoplastic response at multiple sites (liver and kidney) in more than one species (positive response in the rat and mouse) and in more than one sex (female mice and male rats). Thus, taking into account the overall weight of evidence, the induction of liver tumors in mice by THF may be predictive of a carcinogenic response in humans.

The NTP study presents evidence of THF as a potential human carcinogen. The draft EIR's failure to consider this potential carcinogenicity in evaluating public and worker exposures to THF is contrary to public health regulatory practice and standards.

Multipathway Exposures to Solvent Origin Leachates

The draft EIR has completely ignored exposure to CPVC pipe solvent origin leachate through dermal and inhalation pathways in its health risk analysis. Exposure through these pathways have been shown to be significant in a multitude of studies, some of which have been published since completion of the 1989 draft EIR. Those studies cited in the comments submitted by Smith and Wright on the 1989 draft EIR on behalf of the California Pipe Trades Council are not repeated here, but are incorporated by this reference.

- Weisel and Jo (1996) studied dermal and inhalation exposures to volatile organic compounds (VOC's) in human volunteers. The chloroform and trichloroethene concentrations in exhaled breath were elevated in each subject after both inhalation and dermal exposure during showering demonstrating that chemicals in the water entered the body by both routes. Breath concentrations were also elevated after dermal exposure via bathing. In contrast to ingestion, after inhalation and dermal exposure, the exhaled breath had elevated levels for extended time periods, indicating that the compounds were distributed throughout the bloodstream before being metabolized. The expiration data demonstrated that dermal exposure contributes as much to the body burden of chloroform or trichloroethene as inhalation exposure while showering with water containing these contaminants. Extended bathing yielded an even greater dose. It was concluded that for typical activities of drinking and showering, each exposure route contributes similar internal doses and that the total internal dose from a 10 minute shower or a 30 minute bath is greater than that from ingesting 2 liters of water.
- Wilkes et al (1996) evaluated the range of possible inhalation doses taking into account varying activity patterns and water uses. The mean daily potential inhalation dose (PID) predicted in this study ranged from approximately 21 to 39 ug/day. This compared to a PID resulting from drinking between 1 and 2 liters of the same contaminated water. The investigators noted that the 95th percentile value for each group was much higher,

ranging from approximately 70 ug/day to 103 ug/day. The equivalent PID would be reached by drinking 3.5 to 5 liters of water, respectively.

- Experiments were performed in an intact unoccupied house in an attempt to quantify the transfer of pollutants (trichloroethylene) from tap water in showers to indoor air (McKone and Knezovich, 1991). The results of this investigation suggest that inhalation exposure in showers could be equivalent to ingestion of 1 to 4 liters of tap water.
- A three compartment model was used to estimate inhalation exposure to VOC's released from all water uses indoors (McKone 1987). The model was applied to VOC's including chloroform detected in California water supplies. Estimates of the ratio of inhalation uptake to ingestion uptake ranged from 0.8 to 6.
- Measurements of air concentrations of trichloroethylene (TCE) in showers using TCE contaminated drinking water showed increases with time to as high as one third of the occupational TLV (Andelman, 1985).
- Giardino and Andelman (1996) characterized the emissions of three VOC's commonly found in drinking water (TCE, chloroform, and the pesticide DBCP) in a experimental shower and found that the temperature of the water constituted the major effect on the rate and extent of volatilization. Other factors include nature of the volatilizing chemical, air and water flow rates, and nature of the water use (e.g., bath versus shower). The extent of volatilization for such VOC's as chloroform ranged from 50% to 90% (Andelman, 1990).

As illustrated by the results of the aformentioned studies, the exclusion of dermal and inhalation pathways of exposure leads to a significant underestimation of the potential health risks from solvent origin leachates. Consequently, it is not possible to conclude that no significant health impacts would result by simply comparing the levels of solvents determined from the leaching studies with the MDWL's and STEL's based on oral ingestion alone.

Synergistic and Additive Effects of Solvent Leachate

The draft EIR's assertion on page 38 that "In the cases of the solvents used in CPVC primers and cements, there is no evidence to suggest synergistic effects between the four solvents" is misleading. The draft EIR has failed to present and consider in its analysis the evidence demonstrating the ability of MEK, acetone and possibly THF to potentiate the toxic effects of other chemicals including common contaminants of tap water. Potentiation of toxicity, particularly due to acetone and MEK, would be expected to be significant since even an increase of naturally occurring ketones in the body via diabetes can precipitate potentiation (WHO, 1993).

The principal toxic effects associated with exposure to MEK are its ability to potentiate the known toxicity of other solvents (WHO, 1993). A multitude of studies in experimental animals have demonstrated MEK's capacity to enhance liver and kidney toxicity of the haloalkanes. The most studied interactions have been with carbon tetrachloride and chloroform. In rats, at doses used, MEK and the haloalkanes separately produced mild liver and kidney injury at most. When exposure to MEK was followed within 10 to 48 hours by a haloalkane, there was severe injury to the liver, with marked and abrupt replacement of normal hepatic cells by necrotic and fatty vacuolated tissue (Hewitt et al, 1986). A dose as small as 0.072 g/kg potentiated the affects of chloroform given 18 hours later. Lower doses were not studied.

While acetone itself is only moderately toxic, it potentiates the toxicity of a variety of chemicals (ATSDR, 1994). The best studied interactions are with carbon tetrachloride and chloroform. One of the most studied effects of acetone is its ability to induce microsomal enzymes (ibid). Acetone induces its own metabolism by this mechanism and potentiates the toxicity of numerous other chemicals by enhancing their metabolism to reactive intermediates. This induction has been documented in many species and therefore poses a concern for humans exposed to acetone and the chemicals whose toxicity is potentiated by acetone (ibid). Charbonneau et al, (1991) studied the effect of acetone on the severity of liver injury caused by haloalkane mixtures. The results showed first that the individual haloalkanes were not hepatotoxic when administered alone to rats at the doses tested. Acetone pretreatment, however, potentiated liver injury of chloroform, carbon tetrachloride, 1,1-dichloroethylene, and 1,1,2-trichloroethane at these same doses. Hewitt and Plaa (1983) demonstrated that the potentiation of 1,1,2-trichloroethane and 1,1,-dichloroethylene hepatotoxicity by acetone followed a biphasic dose response curve. Low dosages of acetone increased the liver toxicity of these chemicals whereas higher dosages were without effect or in some cases protected against hepatotoxicity.

Chloroform induced nephrotoxicity is potentiated by pretreatment with MEK and acetone (Hewitt and Brown, 1984; Raymond and Plaa, 1995). Brown and Hewitt (1984) demonstrated that MEK and acetone produced a dose-related potentiation of chloroform liver and kidney injury. The relationship between ketone dosage and magnitude of potentiated response was nonlinear. Maximum potentiation of chloroform toxicity occurred at lower dose ranges. Greater dosages were associated with a reduction in the degree of chloroform injury.

THF has been shown to enhance the toxic action of a number of compounds, in particular by stimulating increased absorption of reactive metabolites (Moody, 1991). While there is little evidence to suggest that it would be a direct hepatotoxin at relatively low doses, THF's ability to inhibit drug metabolism reactions and enhance the absorbance of reactive metabolites may potentiate a hepatotoxic response (ibid). Hepatotoxicity is one of the more consistent responses noted in acute and chronic exposures to THF (ibid).

Based on the results of the aforementioned studies, adverse health effects from co-exposure to MEK, acetone or THF and other common contaminants of drinking water would not be unexpected.

Health Effects of Organotins in Leachate

The conclusion that pipe origin leachate in the form of organotins is not likely to cause adverse health effects or significant environmental contamination is not substantiated by any published data and references from the literature. As discussed previously in the section on NSF standards, the MAL's on which the draft EIR's conclusions are based are not adequately health-protective.

The draft EIR's statement on page 40 that "the organotins used as stabilizers in CPVC are far less toxic than TBTO" is misleading and inaccurate. The draft EIR has ignored the potential health effects of exposure to diorganotins, particularly those studies published since the 1989 draft EIR. A brief summary of this information is presented below. It is also important to note that little data are available regarding the potential health effects of long term exposure to organotins.

The target organs of exposure to organotins are the central nervous system, skin, liver and bile duct, immune and reproductive systems (Snoeij et al, 1987; WHO, 1980). There are few data regarding the toxicity of mono organotins but they are believed to be the least toxic of this class of compounds (Hunt and Wilkinson, 1990; WHO, 1980). As discussed in the draft EIR, triorganotins are highly toxic to the central nervous system. Both di and triorganotins are irritants to the skin and eyes of animals and man, and are powerful metabolic inhibitors (Snoeij et al, 1987; WHO, 1980). With regards to their effects on the liver and bile duct, immunotoxicity, and reproductive and developmental toxicity, diorganotins are the most toxic of organotins tested (Ema et al, 1995; Seinen et al, 1977; Snoeij et al, 1987, 1988; Ueno et al, 1994).

Diorganotins are hepatotoxic (Seinen et al, 1977; Snoeij et al, 1987; WHO, 1980). Acute exposure to dibutyltin can cause inflammatory changes in the bile duct which can lead to peritonitis and pancreatitis. The latter disease may follow a chronic course (Merkord et al, 1997). DBTC-induced acute pancreatitis in rats is studied as a type of experimental pancreatitis that resembles the human form of this disease (ibid). Necrotic changes have been produced in the livers of mice, rats and rabbits after short term and subchronic exposure to diorganotins (Seinen et al, 1977; Snoeij et al, 1987; WHO, 1980). Ueno et al (1994) compared the hepatotoxicity of mono, di and triorganotins in mice and concluded that dibutyltin dichloride (DBTC) is more hepatotoxic than tributyltin chloride (TBTC).

Diorganotins have a marked selective effect on the immune system, especially t-lymphocytes (Hunt and Wilkinson, 1990; Seinen et al, 1977; Snoeij et al, 1987; WHO, 1980). Dibutyltins cause a dose dependent atrophy of the thymus, spleen and lymph nodes. As a consequence of their selective lymphocytotoxicity, dibutyltins cause immunosuppression especially of cell mediated immunity. T-cell dependent humoral immunity was also decreased upon dialkyltin exposure (Snoeij et al, 1987). Li et al (1982) demonstrated that DBTC impaired antibody formation by lymphocyctes. These effects were observed at doses with low cytotoxicity. Some trialkyltins are also immunotoxic. Tributyltin-induced thymus atrophy is believed to be caused by its metabolite dibutyltin (Snoeij et al, 1988).

Diorganotins are potent teratogens (Ema et al, 1992, 1995; 1996a; Noda et al, 1992, 1993). Administration of dibutytin during early organogenesis resulted in marked embryolethality and teratogenicity. Oral administration of DBTC to pregnant rats through the period of organogenesis resulted in a significant increase in the incidence of fetuses with malformations even at a dose which did not induce maternal toxicity. Embryos were very highly susceptible to teratogenic effects when exposed on days 7 and 8 of gestation.

Ema et al (1995) compared the developmental toxicity of butyltin trichloride, DBTC, and TBTC in rats during the susceptible period for the teratogenesis of DBTC. Treatment with DBTC resulted in a significantly lower maternal weight gain, lower fetal weight and higher postimplantation embryolethality. A significant and markedly increased incidence of fetuses with malformations such as exencephaly, cleft jaw, cleft lip, ankyloglossia (abnormal shortness of the frenum of the tongue), club foot, deformity of the vertebral column in the cervical and thoracic regions and of the ribs, and ano or microphthalmia (abnormal small size of the eyes) were observed. TBTC was found to be embryotoxic but not teratogenic.

Noda et al (1993) studied the teratogenic effects of various dibutyltins and found that the di-*n*-butyl group rather than the anionic group was important in the teratogenicity of dibutyltin compounds as well as in the other kinds of toxicities. Although the incidence of fetuses with malformations was different, the number of dams with external or skeletal malformed fetuses were similar within these organotin treated groups. Therefore, there may be no significant difference in the teratogenic potency among the various dibutyltin compounds.

Ema et al (1996b) demonstrated that *in vitro* exposure to DBTC interferes with normal development of embryos during 3 different stages of organogenesis in a dose dependent manner, and that susceptibility to embryotoxicity including the dysmorphogenic potential of DBTC varies with developmental stages of the embryos. Embryos during earlier stages appear to be much more sensitive to *in vitro* exposure than during late stages. These results are consistent with Ema et al (1996a) where the developmental toxicity of DBTC was manifested only by a significant decrease in weight of fetuses during late organogenesis. In contrast, administration of DBTC during early organogenesis resulted in marked embryolethality and teratogenicity. Noda et al (1994) found that dibutyltin is transferred to embryos, and embryonic levels of DBTC exceed those in maternal blood suggesting that rat embryos may be able to accumulate DBTC.

Rat embryo limb bud cell culture system has been employed as a teratogenicity screen on the assumption that chemicals which have specific action on cell differentiation are likely to have teratogenic potential (Yonemoto et al, 1993). Except for monobutyltin, the organotins tested were very strong inhibitors of cell differentiation and cell proliferation compared with teratogenic metal compounds. The results of this study suggest that dibutyltins have the highest teratogenic potential (ibid).

As presented in the section on NSF standards, Noland et al (1982) demonstrated that monomethyltin can induce learning deficiencies in young rats. Exposure of rat pups to monomethyltin chloride via their dams drinking water throughout gestation and postpartum for 21 days experienced significant learning deficits and changes in locomotor activity levels. Animals in all dose groups (12, 40, 120 mg/l of tin) displayed some learning disability compared with the controls. Concentrations of organotins in CPVC pipe leachate could exceed a STEL derived from the results of this study (Boettner et al, 1982).

There are no studies of the chronic toxicity or carcinogenicity of organotins in humans and few studies have been conducted in animals (Hunt and Wilkinson, 1990; WHO, 1980; WHO, 1990). The data on the carcinogenicity of organotins is limited to only a few compounds. Some studies have given negative results while others were equivocal (ibid). Ciba-Geigy conducted a 2 year carcinogenicity study of a mixture of octyltins and found a significantly increased frequency of primary tumors of the thymus (Hunt and Wilkinson, 1990). Organotins have been shown to be genotoxic in a variety of *in vitro* and *in vivo* mammalian cell and bacterial assays suggesting that they are potentially carcinogenic

(Hamasaki et al, 1993; Li et al, 1982; Sasaki et al 1993). Dibutyltins were shown to be genotoxic in mammalian cells at concentrations less than 1ppm (Li et al, 1982). Sakaki et al (1993) found that the number of chromosome aberrations in Chinese hamster ovary cells induced by chlorinated tap water extract was increased by post-treatment with organotins.

Based on the results of the aforementioned studies, significant adverse health effects resulting from exposure to diorganotins would not be unexpected. These effects include toxicity to the immune system, liver and bile duct, and the reproductive system. Also, diorganotins are potent teratogens and exposure to mono-organotins *in utero* can result in behavioral effects. Furthermore, the genotoxicity of organotins suggest that they are potentially carcinogenic.

Disinfection By-Products

As stated by the draft EIR, the solvents contained in CPVC pipe cements can leach into drinking water and serve as trihalomethane (THM) precursors. Also, THMs can be formed and/or are used in CPVC manufacture. The document has not provided data to support its claims that the levels of THMs contributed by CPVC pipe, in particular chloroform, would not significantly impact the already exisiting concentrations and health risks of common contaminants of drinking water. Note that the annual status report on drinking water quality in California (CDHS, 1997) and Lam et al (1994) stated that the most frequently observed VOC's continue to be chlorinated hydrocarbons and THMs. Furthermore, over the past years of sampling and testing new sources and chemicals, the number of detections and MCL exceedances has increased proportionally. This trend continued in 1996 indicating that on a statewide basis more detections and MCL exceedances are seen as more new sources are tested (CHDS, 1997).

As discussed in an earlier section of this commentary, acetone and MEK can potentiate the toxic effects of THMs commonly found in tap water and that these effects can occur at low concentrations. Similarly, the potential significance of exposure to THMs through inhalation and dermal pathways has not been considered. Also, although MEK is not chemically reactive under conditions found in most natural waters, water containing free halogens (such as chlorine) or hypohalides can result in the formation of chloroform. The investigators stated that this can be a cause for concern in chlorinated waste water and water supplies since the chloroform thus produced is more toxic than the original MEK (USEPA, 1985).

Chloroform has been shown to be embryotoxic, fetotoxic, and teratogenic in laboratory animals (ATSDR, 1997). Waller et al (1997) found a modest association between tap water containing THMs and spontaneous abortion with the risk beginning to increase at approximately 75 ug/l (70 ppb) THM. Although the study suffered from nondifferential exposure misclassification which would underestimate the observed risks, there was no recall or selection bias. Also, follow up of the cohort was virtually complete. The investigators stated that it is not unusual for concentrations of THMs to exceed 75 ug/L in chlorinated drinking water. Kramer et al (1982) studied developmental effects in humans after exposure to chloroform. The estimated relative risk for intrauterine growth retardation associated with drinking water supplies with chloroform concentrations of greater than 10 ppb was 80% higher than the risks for sources with undetectable levels of chloroform. Drinking water sources with levels of chloroform ranging from 1 to 9 ppb had an elevated risk of 30%. Note that the USEPA's MCL for THMs of 100 ppb is much greater than the concentrations at which effects were reported in each of these studies and that the draft EIR has relied on this standard in its evaluation of potential health risks of disinfection by-products.

Chloroform has been shown to induce liver and kidney tumors in laboratory animals (ATSDR, 1997). Chlorinated drinking water contamination has been consistently linked to gastrointestinal and urinary tract cancer (ATSDR, 1997; Cantor, 1997; Clark and Goodrich, 1986). The results of the epidmiological studies tend to report small relative risks in a range where uncontrolled confounding or other sources of bias might be important. However, some of these studies also suffer from inadequate latency and misclassification of exposure which would tend to underestimate any observed effects (Cantor, 1997). It is important to note that the public health impact of small elevations of risk among large exposed populations, as is the case with exposure to chlorinated drinking water, can be substantial (ibid).

The National Toxicology Program conducted a series of studies in rats and mice exposed via the drinking water to a chemical mixture of 25 groundwater contaminants at concentrations with environmental relevance (NTP, 1993b). Although there was no clear evidence of toxicity in the 26 week study in mice, specific studies of immunotoxicity, myelotoxicity and genetic damage showed adverse effects at concentrations similar to those used in the 26 week studies. The fact that studies with typical toxicological endpoints revealed no significant evidence of toxicity in mice suggests that conventional approaches are inadequate to fully assess more subtle toxicity resulting from long term exposure to low levels of environmental contaminants (ibid).

Although it is shrouded in controversy, the USEPA has proposed to raise the MCL for chloroform to 300 ppb (Risk Policy Report, June 1998). As discussed previously in this section, health effects have been reported from exposure to chloroform at levels lower than the current MCL for THM's of 100 ppb. It is also important to note that raising the level of chloroform in drinking water would result in an increase in the likelihood of toxic interactions between CPVC pipe leachate and chloroform in the drinking water.

The draft EIR does not provide any factual information to substantiate its conclusions that changes in disinfection methods in public water supply systems that will eliminate or at least greatly reduce THM formation will be instituted. Even if this is indeed the case, the draft EIR would need to include an analysis of the data to support the contention that the "other disinfection methods" combined with the use of CPVC pipe for potable water would not result in potential adverse effects.

Depending on the levels of chloroform that would be added to background levels (which are not disclosed by the draft EIR), the chloroform leaching that would be allowed by project approval could result in adverse health effects, including carcinogenic effects. In addition, potentiation of the toxicity of chloroform and other THM's by CPVC leachate would not be unexpected. These issues must be addressed by the draft EIR. The draft EIR's conclusions that there will not be any signifnicant adverse health effects from disinfection by-products is not supported by any analysis or evidence presented in the document.

Threshold Limit Values (TLV's) and Worker Health and Safety

The statements in the draft EIR that TLV's are set to protect the health of workers over their lifetimes with a substantial margin of safety and that TLV's are based on NOAELs are inaccurate. The ACGIH states that TLV's "refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect" (ACGIH, 1988). However, current medical reports regularly show that work-related illness and death occur in workers exposed to toxic substances at levels lower than the TLV's and that these values are often chosen to justify long existing levels of exposure to toxic substances in industrial settings with little relationship to the protection of worker health and safety (Roach and Rappaport, 1990; Tarlau, 1990; Ziem and Castleman, 1989).

In their examination of the development of TLV's, Ziem and Castleman (1989) found that no complete literature searches were done, much medical information was missed, and that the effects of long-term exposure were especially overlooked. For at least 90% of the TLV chemicals, adequate data on chronic effects were unavailable either from studies in animals or of workers with long term exposure to known concentrations of the chemicals of concern.

The New Jersey Department of Health evaluted the chronic health effects data from the EPA's Integrated Risk Information System (IRIS). Workday air concentrations were calculated for noncarcinogens and carcinogens. The resulting exposure limits were frequently three or more orders of magnitude lower than the TLV's (ibid).

Roach and Rappaport (1990) evaluated the published references of human data used to develop TLV's reported in the 1976 and 1986 editions of "Documentation of Threshold Limit Values" by the ACGIH. The results indicated that where the exposure was at or below the TLV, only a minority of studies showed no adverse effects and the remainder indicated that up to 100% of those exposed had been affected. The analysis also revealed that the authors' conclusions in the references regarding exposure-response relationships at or below the TLV's were generally found to be at odds with the conclusions of the TLV Committee.

The following excerpt is from Roach and Rappaport (1990) regarding the references used by the ACGIH to supposedly validate the TLV's for acetone and MEK (also known as 2-butanone):

> Acetone
>
> Effect. "In view of the widespread use of acetone, its volatility and the paucity of reports of illness, it must be considered one of the least toxic of the common solvents. A limit of 1,000 ppm is recommended." (ACGIH, 1976)
>
> Validation? "Acetone produced slight irritation at 300 ppm, but 500 ppm was still tolerated by most of 10 subjects." (Nelson et al, 1943)
>
> 2-Butanone
>
> Effect. "A TLV of 200 ppm should prevent any injurious effects and minimize complaints about odor and irritation." (ACGIH, 1976)
>
> Validation? "Butanone produced slight nose and throat irritation in some of 10 volunteers at 100 ppm." (Nelson et al, 1943)

Since both MEK and acetone produce irritation of the eyes, nose, and throat in human volunteers at at least half the TLV (ATSDR, 1994; WHO, 1993), it is not surprising that a substantial percentage of plumbers reported experiencing irritation during the installation of CPVC pipe (Bellows, 1989). Note, also that not only are these TLV's inaccurate, they do not incorporate margins of safety nor are they based on long term exposure.

Similarly, the draft EIR's reliance on the TLV for THF and its conclusions that there is still no information proving adverse health effects of exposure to THF are not substantiated. The potential carcinogenicity of THF has been discussed in detail in an earlier section of this report. Recall, that the NTP concluded that based on the results of the animal cancer bioassay, the TLV for THF of 200 ppm is too high since tumors were induced in mice at this dose. Similarly, alteration of tracheal ciliary activity was observed in rabbits exposed by inhalation to 100 ppm THF (Ikeoka et al, 1984). Furthermore, the TLV committee itself has questioned the validity of the TLV for THF: "given the epithelial changes in the respiratory epithelium of rats that inhaled 200 ppm 4 hours/day, 5 days/week (Elovaara et al, 1984) and in view of the well-known differences in rodent and human anatomical parameters which contribute to upper airway absorbed dose and the associated response, the margin of safety afforded by the TLV for this substance is currently under review by the TLV Committee." (ACGIH, 1986). Indeed, even one of the manufacturers of THF has recommended a 25 ppm level (see Dupont MSDS).

Bellows (1989) found that the probablity that the TLV's for combined short term exposure to THF, MEK, cyclohexanone, and acetone would be exceeded during at least one 15-minute period over a typical workday was 68%. The estimated exceedance probability was 10% for the full shift exposure limits. Since the TLV's are not health protective, these probabilities are obviously much higher. In air samples representating full shift exposures, the average concentrations of THF and MEK were 47 ppm and 10 ppm, respectively. The highest measured full shift exposures were 158 ppm THF and 45 ppm MEK (ibid). Analysis of exposure to THF via urine samples showed that a significant portion of a worker's total THF dose may be absorbed through the skin. The total THF exposure (airborne and dermal) of these workers were estimated to be equivalent to airborne exposures of 150 to 740 ppm.

Based on these exposure data for a CPVC pipe installation worker and the health effects literature for CPVC cement solvents and primers (in particular THF and MEK) significant adverse health effects would be expected from both short term and long term exposure. Such adverse health effects are even more likely to occur under worst case conditions since the results of Bellows (1989) are not based on a worst-case analysis of exposure. Without taking these considerations into account, there is no scientific basis for the draft EIR's findings of no significant adverse health effects on workers as a result of CPVC solvent exposures.

Noncancer Health Effects of THF

As mentioned in the previous section, the statement in the draft EIR that there are no data proving adverse health effects of exposure to THF is inaccurate. In addition to the results of studies already presented in this document, the draft EIR has also disregarded the following published reports of the toxicity in humans exposed to THF:

- THF has been shown to be irritating to the skin, eyes, and mucous membranes. However, no specific concentrations at which these effects occur has been reported (ACGIH, 1991). Headache, dizziness, fatigue, nausea, and tinnitus have also been reported (Elovaara et al, 1984; Juntunen et al, 1984; Stoughton and Robbins, 1936; Tolot et al, 1968)
- There are two case reports of toxic hepatitis in plumbers exposed to THF (Garnier et al, 1989). Both plumbers had been working in confined spaces repairing plastic pipe with THF-containing glue. One plumber had been working in this situation for eight hours a day for three days, while the other had been working for only a few hours before being admitted to the clinic with symptoms. Note that the liver has been shown to be a target organ after subchronic and chronic exposure to THF in laboratory animals (Chhabbra et al, 1990; 1998).
- Tolot et al (1968) reported that liver enzymes were slightly elevated in a worker 6 weeks after an episode of subacute THF poisoning. Liver enzyme activities were elevated in a man exposed to THF together with other potentially hepatotoxic solvents (chloroform and trichloroethylene) (Edling, 1982). Liver biopsy revealed moderate fatty infiltration. Juntunen et al (1984) also observed a transient rise of liver enzymes in a previously healthy individual exposed occupationally to THF.
- Albrecht et al (1987) reported a case of immunoglobulin-A nephropathy in a plumber fitting PVC pipe in a confined space with short term exposure to THF ranging from 389 to 757 ppm. Small amounts of MEK and cyclohexanone were also present. The investigators suggested that the massive short term exposure may have exacerbated predisposition to renal disease. Note that the kidney has been shown to be a target organ after chronic exposure to THF in rodents (Chhabbra et al, 1998).

Based on the results of the aforementioned studies and the exposure data for a CPVC pipe installation worker as described by Bellows (1989), significant adverse health effects would be expected from both short and long term exposure to THF. Without taking these data into account, there is no scientific basis for the draft EIR's findings of no significant adverse health effects on workers as a result of exposure to THF.

Interaction of Acetone, MEK, THF and Solvent Mixtures

The draft EIR disregards the potential of MEK, acetone and THF to interact with other solvent chemicals. As discussed previously in this document, the principal toxic effects noted with MEK and acetone exposure stem from their ability to potentiate the known toxicity of other solvents and that the exposure limits for these chemicals are flawed since they do not account for these effects.

The World Health Organization has concluded that although there are no reports of MEK potentiation of haloalkanes renal and hepatic toxicity in humans, chronic co-exposure to MEK and either unbranched aliphatic or haloalkane solvents represents a significant potential occupational hazard where serious toxic effects could occur (WHO, 1993). MEK potentiation of hexacarbon neurotoxicity may have caused at least one major industrial accident in which an outbreak of polyneuropathy was followed by introduction of MEK into a solvent mixture (Allen et al, 1974).

Dyro (1978) described three case reports of polyneuropathy in shoe factory workers. The workers had been exposed to MEK and acetone or MEK and toluene at concentrations well below the TLV's for these substances. In one plant, the exposure levels for most cementers were in the range of 10 ppm MEK and 25 ppm toluene. Measurements in the second plant showed concentrations of MEK ranging from 21 to 180 ppm and acetone from 36 to 250 ppm. The current TLV's for MEK and acetone are 200 ppm and 750 ppm, respectively.

Adverse effects of solvent mixtures have been described even in the absence of any known potentiated effects. A PVC pipe fitter who worked in confined spaces using MEK and a glue containing 60% THF exhibited effects on the peripheral nervous system that resolved 2 months after exposure ceased (Viader et al, 1975). An otherwise healthy adult with no history of neurological disease experienced generalized convulsions upon awakening from anesthesia. His occupational history revealed over 10 years of experience with PVC pipe insulation. The investigators concluded that the interactions of THF and enfluran may provoke epileptic seizures. Watery eyes, gastric distress, fainting, convulsions, twitching, headache, and spinal pressure were observed in two cases of acute exposure while waterproofing seams of raincoats with resins containing acetone and MEK (Smith and Mayer, 1944). Juntunen et al (1984) described a case report of cerebral convulsions after enfluran anesthesia and occupational exposure to THF.

The results of studies in Finnish car painters suggested that long term exposure to complex solvent mixtures whose components individually and jointly are far below their concentration limits may produce significant adverse effects (Husman, 1980; Husman and Karli, 1980). Likewise, Noma et al (1988) proposed that complex mixtures of VOC's rather than a high concentration of any single compound may be responsible for unhealthy air in buildings. It should be noted that MEK is widely used as a component of solvent mixtures (WHO, 1993).

Based on the aforementioned studies, adverse health effects resulting from the interaction of CPVC solvents would not be unexpected among exposed workers. Without consideration of the available scientific data as described above, it is not possible for the draft EIR to conclude that no adverse health effects would occur.

Low Volatile Organic Compound (VOC) Solvent Cements and Primers

All assertions regarding the safety of the use of low VOC cement solvents and primers by the draft EIR are not substantiated:

- No data are presented regarding the chemical composition of these new formulations. As a consequence, meaningful comments on the potential health risks associated with their use is impossible.
- No data are provided to verify the claims that low VOC formulations would result in emissions of solvents at levels not likely to produce significant health effects in exposed workers nor that their use will result in less leaching.
- As stated in the draft EIR, low VOC formulations in the form of one step cements have not been approved for use.
- The authors of the draft EIR do not take into account that dermal exposure of workers is still likely to occur in most work settings and that the extent of exposure via the dermal route is significant (Bellows, 1989).
- We are informed that the total chemical content of the low VOC solvent cements and primers has not been reduced (Bellows, 1998). If so, no reduction in health risks would be expected.

The failure of the draft EIR to present data on the chemical composition of low VOC solvent cements and primers precludes meaningful comment on their potential health effects. Without such information, there is no support for the draft EIR's conclusions that no significant health effects would result with their use.

Potential for Long Term Health Effects from Acute Exposure

The draft EIR ignores the potential of long term health effects resulting from acute exposure to CPVC solvent cements and primers. One such long term effect on the respiratory system is referred to as Reactive Airways Dysfunction Syndrome (RADS). RADS is distinguished physiologically by chronic persistent nonspecific airway hyper-responsiveness and usually occurs after single brief high-level exposure to an irritant gas, vapor or fume (Brooks et al, 1985). Without consideration of the potential for chronic effects resulting from high short term exposure to CPVC solvents, there is no support to the draft EIR's conclusions of no significant adverse health effects.

Mitigation Measures

The draft EIR's conclusion that simply following the material labels and the recommendations of the Material Safety Data Sheets (MSDS's) would greatly reduce the potential for exposure to solvents is not supported by any facts or analysis. Based on our analysis of labeling requirements and MSDS recommendations, we find that such measures would not reduce exposures. First, they are too general and generic to serve as reliable or effective safe work standards. Second, these and other "mitigation measures" discussed in the draft EIR do not appear practical and enforceable in actual work settings.

Bellows (1989) found that ninety percent of employees in every type of pipe installation had received no health and safety training. Of 78 plastic pipe workers monitored, only two workers said they had been informed about potential hazards of the substances in CPVC pipe primers and cements. Most workers reported that they had read the warning labels on these products and many found such warnings as "avoid contact with skin" and "use only in well-ventilated areas" to be comically impractical (ibid).

For all the above reasons, we conclude that significant health effects in exposed workers would result from repeated exposure to CPVC cement solvents and primers containing acetone, MEK and/or THF.

Health Effects of Leachate from Copper Pipe

The draft EIR does not provide data to substantiate its conclusions that the potential risks from exposure to copper pipe leachate are greater than the potential risks associated with CPVC pipe leachate. This section of the document does not contain a scientific analysis of either leaching data or health effects information regarding copper pipe from the published literature. What little toxicological data that is presented is frequently discussed without reference to the specific chemicals involved and without citations from the peer-reviewed literature. This is particularly apparent for nonmetallic leachate from fluxes and cutting fluids.

The draft EIR does not provide recent data to support its contentions that current levels of lead leachate from copper pipe are likely to cause adverse health effects. As stated in the document on page 77, after July 1986, the state of California prohibited the use of solders containing more than 0.02 % lead. The information regarding lead exposure that is presented by the draft EIR is cited from studies conducted prior to this date.

The draft EIR has mischaracterized the health effects from exposure to copper. Furthermore, no data are provided to support the assertion that current levels of copper in leachate would result in significant adverse health effects. Unlike the chemicals found in CPVC pipe leachate, copper is necessary for good health (ATSDR, 1990a). Copper has also found its place in pharmacology where it has been used as an anti-inflammatory agent (Garret and Whitehouse, 1987). Deficiency of copper causes abnormal hemoglobin synthesis leading to hypochromic and microcytic anemia (ATSDR, 1990a).

The sudden ingestion of large amounts of copper may lead to acute poisoning (ATSDR, 1990a; Seymour, 1987). However, acute copper poisoning is rare and self-limiting since it induces nausea, vomiting and diarrhea which protect against the more serious systemic effects. Furthermore, one can usually taste copper in drinking water before experiencing adverse effects (ATSDR, 1990a).

The only significant example of copper toxicity in humans is Wilson's disease (ATSDR, 1990a). It is an autosomal recessive disorder that affects normal copper homeostatis and is characterized by hepatic and renal lesions, and hemolytic anemia. There are no studies regarding reproductive effects in humans or animals following oral exposure to copper. However, intrauterine devices (IUD's) containing copper have been used as contraceptive devices in women (ATSDR, 1990a).There are no data to suggest that copper is carcinogenic in animals or man (ibid).

Infants exposed to high levels of copper in the drinking water may have harmful health effects at lower levels than adults because the homeostatic mechanisms for clearing copper from the body and preventing its entry via the intestine have not yet been developed (ATSDR, 1990a). Two infant siblings who consumed copper for about 9 months in their drinking water exhibited harmful liver effects (Mueller-Hoecker et al, 1988). The current USEPA MCL for copper of 1.3 ppm is not adequately health protective for infants and children under 10 years of age (Sidhu et al, 1995). A MCL of 0.3 ppm (300 ppb) has been proposed that would adequately protect the health of infants, children and adults (ibid). This level would also provide approximately 26% of the nutritional requirement of copper in the diet (ibid). Note that the median and mean levels of copper found in California drinking water are 90 ppb and 167 ppb, respectively (CDHS, 1997).

Based on the aforemention discussion, we conclude that the draft EIR has not scientifically supported its claims that the potential health risks from exposure to current levels of copper and lead from copper pipe leachate are greater than the potential risks associated with CPVC pipe leachate.

Copper in the Environment

The draft EIR has mischaracterized the data regarding the environmental impacts of copper. Copper in natural waters is predominantly found in the Cu (II) state (ATSDR, 1990a). The chemical conditions in most natural waters are such that even at relatively high concentrations, the combined processes of complexation, adsorption, and precipitation control the level of free copper in water and can reduce it to extremely low values (ibid). Furthermore, secondary stage treatment of municipal sewage has been shown to remove on average 49% to 82% soluble copper and 83% to 90% total copper (Aulenbach et al, 1987; Stephenson and Lester, 1987). Also, the production of copper and brass releases relatively little copper to water (ATSDR, 1990a). Copper sulfate is added to lakes, reservoirs, and ponds for controlling algae. However, the levels of copper usually return to pretreatment concentrations within a few days (Perwack et al, 1980).

It is important to note that the draft EIR includes absolutely no discussion or presentation of any data from the scientific literature regarding environmental contamination from the use of CPVC pipe and its potential effects on ecosystems. Consequently, it is not possible for the draft EIR to conclude that the environmental impacts based on the use of copper pipe would be greater than the risks associated with the use of CPVC pipe.

Antagonistic or Competitive Effects of Metal Pipe Leachates

The draft EIR mentions the possibility of synergistic and additive effects of exposure to copper pipe leachate without substantiation with data from the published literature. On the otherhand, the draft EIR, has ignored the results of studies that have found antagonistic effects resulting from co-exposure to metal pipe leachate (i.e., cadmium, copper, zinc). For example, increasing the intake of zinc in the diet has been demonstrated to protect against the onset of copper toxicity in animals (Allen, 1987). Both zinc and cadmium have been shown interfere with copper absorption and metabolism by competing for common binding sites on metallothionein (ATSDR, 1990a).

Other Metals Leaching from Metal Pipe

No data have been provided to support the implication that levels of cadmium or zinc in leachate from metal pipe used to convey potable water would result in adverse health effects. The draft EIR states on page 78 that zinc is found in relatively high concentrations in water conveyed by galvanized steel pipe yet does not mention that zinc, like copper is an essential element in living organisms and is present in all foods (ATSDR, 1993). Zinc compounds are also found in many pharmaceutical products such as sun blocks, diaper rash ointments, deodorants, athletes foot preparations, acne and poison ivy medications, and antidandruff shampoos (ibid).

Deficiencies of zinc can lead to loss of appetite, a decreased sense of taste and smell, depressed immune function, slow wound healing, and male reproductive damage (ATSDR, 1993). Too little zinc in pregnancy may result in growth retardation and depressed mental function.

Large doses of zinc could cause adverse health effects. Acute exposure can cause gastrointestinal problems including stomach cramps, nausea and vomiting (ATSDR, 1993). Chronic effects resulting from long term exposure to zinc include anemia, damage to the pancreas, and decreased levels of high density lipoprotein (HDL) cholesterol.

The draft EIR states on page 79 that zinc levels in copper and steel pipe leachate range from 29-214 ppb and 232-1279 ppb, respectively. The National Academy of Sciences' (NAS) Recommended Daily Allowances (RDA's) of zinc for infants and children are 5 mg/day and 10 mg/day, respectively (ATSDR, 1993). Assuming a 1 liter consumption of tap water per day would result in intakes for zinc of 5 ppm for infants and 10 ppm for children. Note that the leachate levels from both forms of pipe are much lower than the RDA's for both infants and children.

Long-term oral exposure to cadmium may result in toxicity to the kidneys, bone and gastrointestinal tract (ATSDR, 1998a). Based on limited data, exposure to cadmium via ingestion does not appear to be carcinogenic in animals or man (ibid). The USEPA has set a MCL of 5 ppb for cadmium in drinking water (ibid). The draft EIR states on page 79 that cadmium leachate from steel and copper pipe ranges from 0.26-1.13 ppb and 0.02-0.21 ppb, respectively. Note that the cadmium concentrations in copper pipe leachate are 25 to 250 times lower than the USEPA's MCL for cadmium.

In summary, the draft EIR does not provide data to substantiate the suggestion that levels of zinc and cadmium leaching from steel pipe used to convey potable water would result in any significant adverse health effects.

Nonmetallic Leachates from Metal Pipe

The draft EIR does not provide data to support the assertions that levels of non-metallic leachates from metal pipe might pose a potential health risk. Furthermore, it has not been possible to adequately comment on what little toxicity data is presented in this section since the components of the fluxes and cutting fluids which comprise non-metallic leachate have not been provided in the draft EIR or its technical appendix.

The conclusions of the draft EIR regarding the potential health effects of exposure to soldering fluxes appear to be based on the results of Nikora et al (1998). In this study, laboratory simulated soldering experiments of metal plumbing were conducted. The investigators stated that their results demonstrated the release of potentially toxic organic vapors. However, it is important to note that the study concluded that "many of the identified materials appear to be released at what would be considered a measurable level". Other chemicals were said to be released at trace amounts. Based on the results of this study, it is impossible to conclude that advserse health effects would result from such low exposure to the components of soldering fluxes. Furthermore, no data are provided that would indicate their presence in leachate.

We conclude that the draft EIR has not presented data to substantiate any potential exposure to or any potential health risks resulting from non-metallic pipe leachate.

Worker Health and Safety in the Existing Environment

The draft EIR does not provide data to substantiate its conclusions that the potential risks from working with existing piping materials are greater than the potential risks associated with CPVC pipe installation. As was the case in the section on copper pipe leachate, this portion of the document does not contain a factual presentation or analysis of either worker exposure data or health effects information from the published literature. What little health effects data that is presented is frequently discussed without reference to the specific chemicals involved and without references from the peer-reviewed literature.

The statement on page 94 that "toxic and carcinogenic smokes and vapors are produced during soldering operations and released into the workplace atmosphere" is misleading. Again, the draft EIR has relied on the results of Nikora et al (1998) as a basis for its conclusions. As stated in the previous section, this study concluded that only "measurable" or "trace" amounts of substances were identified. Copper pipe solders are composed mainly of tin with small amounts (less than 10% in all) of copper, silver or antimony (Bellows, 1989). The results of Nikora et al (1998) did not reveal the presence of tin, antimony, or lead in the respirable smoke particles. Metal fume monitoring of plumbers during copper pipe installation showed that the average 8-hour time-weighted average (TWA) exposures for copper, tin, silver, antimony and lead ranged from 0.2% to 4% of the exposure limits for these metals (Bellows, 1989).

Furthermore, the statement on page 44 that soldering fumes are carcinogenic is incorrect. There are no studies conducted in animals or humans in the published literature that have provided evidence of the carcinogenicity of exposure to soldering fumes. Of the components of copper pipe solders, there is no evidence of the carcinogenicity of copper, tin, or silver in animals and man, and equivocal evidence of the carcinogenicity of antimony and lead (ATSDR, 1990abc; ATSDR, 1992; ATSDR, 1996; ATSDR, 1998b; USEPA, 1993).

The statement by the draft EIR on page 94 that "tin has produced tumors in experimental animals but is not considered to be carcinogenic" is misleading. The NTP conducted a carcinogenesis bioassay for stannous chloride in rats and mice exposed in the diet. Although some tumors were induced in rats and mice, the NTP concluded that the incidences of tumors relative to the historical control rat and mouse data were similar and not clearly related to stannous chloride administration (ATSDR, 1992).

We conclude that the draft EIR does not provide any scientific evidence to support its conclusions that health effects resulting from working with existing piping materials are greater than the potential risks associated with CPVC pipe installation.

Discussion of Chemical Hazard Evaluation in Appendix B

The risk assessment process as described in this section is a biased and unbalanced commentary that should be indicated as the opinions of the preparers of the draft EIR. The risk assessment process is deliberately designed to be health conservative. It is conservative because it is intended to protect the public health including sensitive members of the population. Sensitive individuals generally include children, pregnant women, the elderly, persons with existing diseases, and individuals with lower levels of protective biological mechanisms due to genetic variabililty within the population. Assumptions made are meant to err on the side of public health protection but in many cases are reflective of an average individual (CAPCOA, 1993).

While uncertainties are inherent in the risk assessment process, these are not as one-sided as implied by the draft EIR. Sources of uncertainty that could lead to an underestimation of risk include exposure through more than one pathway, and the possibility of additive, synergistic and/or potentiated health effects which are generally not accounted for using standard risk assessment methodology.

The statement on page 50 that "exposure assumptions for workers are different from those that are applied to the general public, and allowable exposure limits are generally greater" is misleading. The exposure limits are generally greater because they usually refer to short term high level exposures while exposure limits applied to the general public are intended to protect against chronic effects resulting from long term low level exposure to toxic substances. Other mischaracterizations of workplace standards by the draft EIR have been discussed in an earlier section.

Conclusion

In summary, we find that the conclusions of the draft EIR that no significant health effects would be associated with the use of CPVC pipe for the distribution of potable water are almost entirely unsupported by the scientific literature. In addition, the draft EIR has failed to disclose the toxicological data and studies which support the NSF standards on which it relies to evaluate the significance of the leaching impacts. The absence of this information precludes any meaningful comment on the established NSF standards. Furthermore, as discussed, the published literature indicates that the Maximum Allowable Limits relied upon by the draft EIR are too high to be adequately health protective.

The draft EIR has failed to adequately present and evaluate available health effects data as discussed in this commentary, and have thereby seriously underestimated the potential health impacts of the extended use of CPVC pipe to both exposed workers and residents. Based on our analysis, we find that significant adverse health effects on workers and drinking water consumers from the proposed CPVC approval would not be unexpected.

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